Research and Reviews of Infectious Diseases

ISSN: 2643-6051

Research Article DOI: 10.36959/719/560

Infectious Vulvovaginitis in Adult Women in a Hospital in Dakar

Assane Diop^{1*}, Doha Amrani Joutei¹, Mame Tene Ndiaye¹, Valiollah Abbaspour¹, Saer Diadie¹, Boubacar Ahy Diatta², Maodo Ndiaye², Moussa Diallo², Moctar Mboup³, Mansour Niang⁴, Cheikh Tidiane Cissé⁴, Suzanne Oumou Niang² and Fatimata Ly¹

Summary

Introduction: Vulvovaginitis is mainly infectious. Some of these infections are sexually transmitted.

Objective: Studying epidemiological, clinical, etiologic and therapeutic aspects of infectious vulvovaginitis (IVV) at Institute of Social Hygiene Hospital (IHS) in Dakar.

Materials and methods: This is a cross-sectional and prospective study. It was carried out during a period from 1 March to 31 August 2017 (6 months), at the IHS hospital in Dakar. All patients with VVI and an age of 18 years or more were Included.

Results: We included 177 patients, a hospital frequency of 5.7%. The mean age was 33.68 years. 71.8% of the patients were married, and 9% had no sexual activity before. Sexual intercourse was only vaginal in 57% and genito-anal in 8.8%. Genital pruritus was present in 75% of cases and leucorrhoea in 73.4%. The symptoms were associated in 81.4% of the cases. Vulvitis was found in 28.2% (n = 50) of cases and vaginitis in 83.8% (n = 135) of sexually active patients. In the latter, cervicitis was noted in 52.2%. Condyloma existed in 30%. A vulvar specimen was taken in 75% of the virgin patients, these patients showed no STIs. Cervicovaginal sampling was performed in 60.2% of sexually active patients, in these patients, the infection were as follows: Bacterial vaginosis (62.9%), candidiasis (50.5%), vaginitis with common germs (26.8%), chlamydiosis (14.4%), trichomoniasis (13.4%) and gonorrhea (5.6%). These findings were associated in 56.7% (n = 55) with common-cell vaginitis which was related to the use of intravaginal Herbs (p = 0.00001502) and the genito-anal ratio (p = 0.005596). The etiologic treatment was more effective than the syndrome treatment (p = 0.0002426).

Conclusion: Infectious vulvovaginitis is relatively frequent in adult women. Infectious agents are mainly non STIs. But they are still a public health problem.

Keywords

Vulvovaginitis, Bacterial vaginosis, STIs

Introduction

Vulvovaginal infections mainly occur as vulvovaginitis [1,2]. A few are sexually transmitted (STI), but others are not [2-4]. In non-sexually transmitted vulvovaginitis [4,5], the infectious agent can be a commensal, opportunistic or exogenous microorganism [6]. Vulvovaginitis du to *Candida Albicans* (*C. albicans*) or other Candida can affect up to 75% of women at least once in their lifetime [7]. Bacterial vaginosis due to a very different infectious agent [8] like (*Gardenerella vaginalis* (*G. vaginalis*), *Prevotella sp, Bacteroides sp, Mobiluncus sp, Mycoplasma hominis* (*M. hominis*) and *Ureaplasma urealyticum* (*U. urealyticum*)) is by far the most common cause of vaginal discharge in childbearing women [9]. Vag-

initis secondary to STIs, are the consequence of cervical infections. It is most often the following sexually transmitted germs: *Trichomonas vaginalis* (*T. vaginalis*), *Neisseria gonorrheae* (*N. gonorrheae*), *Chlamydia trachomatis* (*C. tracho-*

*Corresponding author: Assane DIOP, Department of Dermatology/STI, Hospital Institute of Social Hygiene of Dakar, Senegal, Tel: +221-77-634 -76-77

Accepted: January 21, 2020

Published online: January 23, 2020

Citation: Diop A, Joutei DA, Ndiaye MT, et al. (2020) Infectious Vulvovaginitis in Adult Women in a Hospital in Dakar. Res Rev

Infect Dis 3(1):32-39

Copyright: © 2020 Diop A, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.



¹Department of Dermatology/STI, Hospital Institute of Social Hygiene (IHS), Dakar, Senegal

²Department of Dermatology, Hospital Aristide Le Dantec (HALD), Senegal

³Bacteriology Laboratory, Hospital Institute of Social Hygiene (IHS) of Dakar, Senegal

⁴Department of Obstetric-gynecology Hospital Institute of Social Hygiene (IHS) of Dakar, Senegal

matis) and Mycoplasma genitalium (M. genitalium) [10,11]. Herpes virus 1 and 2 are other sexually transmitted infections which can cause vulvovaginitis. Human papillomavirus (HPV) serotypes 6 and 11 are the cause of condylomas in 70 to 100% of cases [12]. STIs are a public health problem in industrialized countries and especially in developing countries [13]. The serious gyneco-obstetric and neonatal complications they cause make their prevention and management particularly important [11,14]. In Senegal, to our knowledge, there is no studies of the etiologies of vulvovaginitis in dermatology department.

The objective of our study was to determine the epidemiological, clinical, etiological and therapeutic aspects of infectious vulvovaginitis in dermatology and gynecology-obstetrics departments in Dakar Institute of Social Hygiene Hospital.

Materials and Methods

It was a descriptive and analytical study with a prospective data collection. It was carried out over a period of 6 months (1 March to 31 August 2017) in the dermatology/STI and gynecology-obstetrics departments of the Dakar Institute of Social Hygiene Hospital.

All 18 years or older women who came to dermatology or gynecology and obstetrics department of Dakar Institute of Social Hygiene Hospital during the study period were included. Clinical signs suggestive of vulvovaginitis were leucorrhoea, dyspareunia, vulvar pruritus, pelvic pain, inflammation of the vulva and/or vagina, and the existence of venereal vegetations. A pre-established inquiry form was used to collect sociodemographic, clinical, paraclinical, therapeutic and evolutionary variables.

The vulva was examined in all patients, and the vagina was examined only in patients with sexual activity. Patients with non-infectious vulvovaginal disease, official sex workers and non-consenting patients were excluded. In the included patients, vulvar or cervico-vaginal specimens were requested to detect the infectious agent.

The bacteriological and parasitological study of vulvar and cervicovaginal samples and serodiagnosis were carried out in the bacteriology laboratory of the Hospital Institute of Social Hygiene (IHS) in Dakar. In case of pregnancy or STIs, syphilitic and retroviral (HIV) serologies as well as HBsAg were systematically requested.

The search for *T. vaginalis* was performed by direct examination. That of *N. gonorrheae*, Candida, and common germs was done by direct examination and culture. *C. trachomatis* was demonstrated by immunochromatography. However, the search for *Mycoplasma genitalium* was not performed because of a lack of reagents.

The syndromic management (empiric therapy) of vulvovaginitis was carried out, after the local samples, according to the algorithm of management of the vaginal discharges. The aim was to treat bacterial vaginosis and candidiasis in the absence of cervicitis. In the case of failure (for example the persistence of signs one week later), trichomoniasis was treated. In the presence of cervicitis, it was necessary to treat trichomoniasis, gonorrhea and chlamydiosis.

Each patient was revisited 7 days after syndromic treatment for clinical evaluation of efficacy; and this treatment was readapted, if necessary, according to the sensitivity of the isolated germs; other check appointments, on day 14 and day 28, were requested.

Patients who did not follow their appointments were attached to the phone to collect the evolving data.

According to the national empiric therapy approach:

- The treatment of gonorrhea consisted of a single dose of 500 mg Ciprofloxacin; in the second line, Cefriaxone is used at a dose of 250 mg in a single injection.
- Chlamydia is treated with Doxycycline 200 mg twice daily for 7 days or Azithromycin 1 g once. In case of allergy to cyclins or for the management of a pregnant or breastfeeding woman, erythromycin 500 mg 4 times daily for 7 days was used.
- Trichomoniasis and bacterial vaginosis are treated with Metronidazole 500 mg twice daily for 7 days or 4 tablets taken once. In pregnant women, this treatment is given after the first trimester.
- Candidiasis was treated by 2% vaginal cream of Clotrimazole: 1 application per day for 3 days, Fluconazole 150 mg single dose (against indicated in pregnant or lactating women) or Nystatin 100000UI: 1 vaginal tablet in the evening at bedtime for 14 days.

The condylomas were treated with one to two sessions of cryotherapy per week.

All partners of patients with sexually transmitted infection had received adequate therapeutic prescription. The study was approved by the Dermatology-Venerology resident training board of Senegal. The members of the board consist of 5 Professors, an Associate Lecturer and 3 Assistants.

Patients were free to participate or not in the study, and to withdraw at any time. Confidentiality was ensured using the initials of their surname and first name. These initials appeared on all documents and databases.

The variables were entered using the Excel 2013 software and analyzed using the IBM SPSS statistics Base 22.0 software. The Chi-square or Fisher test was used to determine statistical differences. This difference was statistically significant if p < 0.05. The strength of the relationship was specified by the Odds Ratio (OR), with a 95% confidence interval.

Results

182 patients were received and 177 were included. The 5 patients did not want to be included. During the study period, the Dermatology and Gynecology and Obstetrics Departments received 2145 and 1005 women aged 18 and over respectively. The hospital frequency of infectious vulvovaginitis was 5.6% (177/3150). It was 4.75% (102/2145) in dermatology-venereology department and 7.46%

(75/1005) in gynecology obstetrics and department. The mean age of our patients was 33.68 ± 11.15 years, with extremes of 18 and 70 years. Patients aged 18 to 44 accounted for 84.7% (n = 150) of the workforce. Patients from the Dakar region accounted for 96% (n = 170) and those from other regions 4% (n = 7). Brides accounted for 71.8% (n = 127) of the cases, and unmarried women accounted for 28.2% (n = 50). Women married with monogamous men accounted for 29% (n = 37) and those under polygamous 71% (n = 90). Of the 51.4% (n = 91) of the patients who were working, the 60.4% (n = 55) were active in the informal sector while 39.7% (n = 36) were in the formal sector. The socioeconomic level was considered low in 56.5% (n = 100) of patients, mean in 40.7% (n = 72) and high in 2.8% (n = 5).

Diabetics accounted for 4.5% (n = 8) of patients, and 40.7% were overweight or obese (n = 72). HIV infection and long-term corticosteroid therapy were found in 2 cases, and systemic disease in 1 case.

There was a previous history of genital infections or sign of genital infections in the last 12 months in 107 patients (60.5%). Sixteen patients, (9%) had never had sex. Table 1 shows the distribution of patients by medical history and lifestyle.

Compensation for intercourse was noted in 12% (n = 6) of unmarried patients. In these unmarried patients, condoms were routinely used in 10% (n = 5) and inconsistently in 84% (n = 42).

Vulvovaginitis was the reason for consultation in 70%

(n = 124) of patients and in 30% was found haphazardly (n = 53). Genital pruritus was present in 75% (n = 133) of patients. Leucorrhea, dyspareunia and pelvic pain were seen in 73.4% (n = 130), 64% (n = 103) and 31% (n = 55) respectively. The average delay period between appearing of the symptoms and consultation time for leucorrhea was 6.5 months (1 day to 8 years). Vulvitis was found in 28.2% (n = 50) of the patients. Speculum examination in sexually active patients showed vaginal discharge in 57, 62% (n = 102) of the patients Vaginitis was present in 83.8% (n = 135). A combination of vaginitis and vulvitis was seen in 20.5% (n = 33) of the patients. A cervicitis was observed in 52.2% (n = 84) of the patients in sexually active patients and the cervical secretion was in 15.8% (n = 28). No signs of herpetic vulvo-vaginitis were noted. Condylomas were found in 30% (n = 53) of the patients. Genital localization with 86.8% (n = 46) and genito-anal localization in 13.2% (n = 7). They were isolated, but with a notion of leucorrhea, in 41.5% (n = 22) of the cases. In clinical examination, 58.5% (n = 31) of them were associated with vulvitis and/or vulvovaginitis.

A vaginal smear was requested for 161 sexually active patients but just 109 patients (61.6%) had done it. In 12 virgin patients we did just vulvar sampling, and cervicovaginal in 60.2% (n = 97).

Vulvar samples revealed $\it C. albicans$ as only germ in 66.7% (n = 8) and associated with other germ in 58.3% (n = 7) of the patients.

Regarding cervicovaginal samples, showed C. albicans as the only germ in 47.4% (n = 46), and in association with other

Table 1: Distribution of 177 patients with vulvovaginal infections by medical history, and lifestyle.

Medical history and lifestyle		Number of people	Percentage (%)	
Obstetric history	Miscarriage	25	15.5	
(No.161)	Stillborn	9	5.6	
	Premature	1	0.6	
	Normal childbirth	126	78.26	
	Diabetes	8	4.5	
	Overweight/Obesity	72	40.7	
	Clobetasol-based bleaching agents	42	23.7	
	Menopause	27	15.3	
Background (No.177)	Immunodepression	5	2.8	
	Current or recent use of antibiotics	30	17	
Background (No.161)	Herbal extracts (powder or liquid) intravaginal	20	12.4	
	Pregnancy	39	24.2	
	Excessive genital hygiene (vaginal shower)	52	32.3	
	Estrogen-progestatory hormonal contraception	9	5.6	
	Endo-uterine gesture (IUD pose)	4	2.48	
Type of sexual intercourse (No.161)	Vaginal	92	57	
	Vaginal, Oral	55	34.2	
	Vaginal, Oral, Anal	11	6.8	
	Vaginal, Anal	3	2	

 Table 2: Different germs found in 12 virgin patients and 97 sexually active patient with vulvo-vaginitis.

Sampling site	Germs	Number of people	Percentage%
Vulva	Candida albicans	8	66.7
(No.12)	Gardnerella vaginalis	8	66.7
	Streptococcus (strepB)	2	16.7
	Escherichia coli	1	8.3
	Germ Association	7	58.3
	Candida albicans	46	47.4
	Gardnerella vaginalis	32	33
	Mycoplasma hominis	19	19.6
	Chlamydia trachomatis	14	14.4
	Streptococcus (strepB)	13	13.4
Corvice vaginal comples	Trichomonas vaginalis	13	13.4
Cervico-vaginal samples (No.97)	Ureaplasma urealyticum	10	10.3
	Staph aureus	6	6.2
	Escherichia coli	5	5.15
	Neisseria gonorrhea	5	5.15
	Klebsiella pneumoniae	2	2
	Candida non-albicans	3	3
	Germ Association	55	56.7

 Table 3: Different etiologies of infectious vulvo-vaginitis in 97 sexually active patients.

Sampling site		Cervical-vaginal sa	Cervical-vaginal sampling	
		number of people	(%)	
Etiologies of vulvo-vagini				
	Vaginosis	20	20.6	
	Candidiasis	16	16.5	
Single agent infections	Chlamydia	2	2	
omgre agent infections	Trichomonose	2	2	
	Ordinary germ vaginitis	2	2	
Mixed infections	Vaginosis - Candidosis	16	16.5	
	Vaginosis - Ordinary Germ Vaginites	6	6.2	
	Chlamydiosis - vaginosis	6	6.2	
	Vaginitis with common germs - Candidosis	5	5.2	
	Trichomonosis - Candidosis - Vaginitis with common germs	4	4	
	Vaginitis with common germs candidiasis - vaginosis	3	3	
	Trichomonose - Vaginosis	2		
	Trichomonosis - Vaginose - Unremarkable germ vaginitis	2		
	Gonococcal disease - Chlamydia - vaginosis	1	1	
	Trichomonose - Candidiasis	1	1	
	Gonococcal - Vaginitis with common germs	1	1	
	Trichomonosis - Vaginites with mundane germs	1	1	
	Vaginosis - Chlamydia	1	1	
	Chlamydia - vaginosis - candidiasis - trichomonose	1	1	
	Candidosis - chlamydia - vaginosis	1	1	
	Common germ shingle	1	1	
	Gonococcie-candidiasis	1	1	
	Chlamydia - vaginosis - candidiasis - gonococcal	1	1	
	Vaginitis with common germs - vaginosis - gonococcal	1	1	

germs in 56.7% (n = 55) of the patient.

Table 2 shows the different germs found in 12 virgin patients and 97 patients in sexual activity.

Bacterial vaginosis was found in 62.9% (n = 619), vagi-

nal candidiasis in 50.5% (n = 49), non-viral STIs in 29.9% (n = 29), and bacterial (or common) vulvovaginitis in 26.8% (n = 26) sexually active women. Of the 58.5% (n = 31) of patients with condyloma who had cervical-vaginal swabs, 22.6% (n = 7) had another STI and 61.3% (n = 19) had bac-

Table 4: Different etiologies of infectious vulvo-vaginitis in 97 sexually active patients.

	Sampling site	Vulva	
		number of people	(%)
Etiologies of vulvo-vaginitis			
Single agent infections	Candidiasis	3	25
	Vaginosis	1	8.3
	Ordinary germ vaginitis	1	8.3
Mixed infections	Vaginosis - Candidosis	5	41.6
	Vaginitis with common germs - vaginosis	2	16.6

 Table 5: Relationship between - clinical and epidemiological characteristics and the type of vulvo-vaginitis.

Characteristics	Bacterial vaginosis	Other vulvovaginitis	P-values	OR; IC 95%	
Fluid discharge	37	41	0.006807	3.76 [1.389-10.18]	
Curd-like discharge	6	25			
Foul smelling discharge	14	10	0.03208	2.703 [1.07-6.832]	
Non-nausea secretions	29	56			
Characteristics	Ordinary germ vaginitis	Other vaginitis	P-values	OR; IC 95%	
Unemployed women	20	35	0.01998	2.857 [1.159-7.043]	
Employed Women	9	45			
Intravaginal Herb uses	13	7	0.00001502	9.143 [3.058-27.34]	
No intravaginal Herbs	13	64			
Pregnancy	11	11	0.005216	4 [1.458-10.97]	
Pregnancy-free	15	60			
Anal vaginal intercourse	8	6	0.005596	4.815 [1.479-15.67]	
No genitalia	18	65	1		
Characteristics	Vulvovaginitis in Candida	Vulvovaginitis to other germs	P-values	OR; IC 95%	
Curd-like discharge	29	2	0.0000001	25.89 [5.745-116.7]	
Fluid discharge	28	50			
Characteristics	STS Vulvovaginitis	Vulvovaginitis Non STIs	P-values	OR; IC 95%	
Cervicitis	29	27	0.000001		
No cervicitis	0	41			
Characteristics	Etiological treatment	Syndromic treatment	P-values	OR; IC 95%	
Healing in Day 14	60	83			
Persistence at day 14	0	18	0.0002426		
Characteristics	Condylomata	No condylomes	P-values	OR; IC 95%	
≤33	42	56			
> 33	11	68	0.00002942	4.636 [2.185-9.836]	
Suburbs of Dakar	41	57	0.0001192	4.016 [1.928-8.366]	
Dakar City Centre	12	67			

terial vaginosis. Tables 3 and Table 4 show the different etiologies of infectious vulvovaginitis in 97 sexual and 12 virgin patients.

The retroviral (HIV) and syphilitic (TPHA and VDRL) and HBsAg serologies were done in 54.2% (n = 96) of the patients. These tests were done in 61.8% (n = 60) of sexually active patients who had cervico-vaginal sampling and in 96.5% (n = 28) patients with a STI. The HIV serology was positive in 2 (2%) of the patients, one of them was a non-pregnant woman with gonorrhea and the other had condyloma. HBsAg was positive in a pregnant woman without STIs. Syphilitic serology was negative in all cases.

An empiric therapy of vulvovaginitis was done in all cases and was customized according to the microbial agent and/or susceptibility of the germs in 33.9% (n = 60). In the 101 sexually active patients who received only empiric therapy, a clinical cure was noted in 82.2% (n = 83) at day 14, and in 15.8% a recurrence was seen at Day 28. As a result, the efficacy of empiric therapy was 68.3%.

In bivariate analysis of variance, there was a statistically significant relationship between the genito-anal ratio and bacterial vaginitis, and the existence of a non-viral STI and cervicitis. Table 5 shows the results of the bivariate analysis.

Discussion

The limits of our work lie in the fact that it is only performed in two departments, and that 38.4% of our patients did not have any vulvar or cervicovaginal samples. Despite this, recruitment is done in two reference centers. It is one of Dakar's reference obstetrics and gynecology services and the reference service for STI management. The second was the unavailability of reagents for *M. genitalium* detection.

Despite these limitations, the validity of our study is based on internal and external arguments. The first are corroborated by the frequency of vulvovaginal candidiasis in diabetics, the predominance of curdled secretions in women with candidiasis, the absence of STIs in patients without sexual activity, the presence of cervicitis in all patients who had a non-viral STI, the efficacy of etiologic treatment versus syndromic treatment. The second is based on the higher frequency of candidiasis as a cause of vulvovaginitis [5,15].

The peculiarities of our study are the high frequency of vulvovaginitis in adult women, the important use of intravaginal Herbal extracts, the high frequency of STIs and the significant percentage of failure of syndromic treatment (empiric therapy).

We found a hospital prevalence of infectious vulvovaginitis of 5%. This prevalence is close to the 6% of low genital infections found by Diadhiou, et al. [16], in adult women in gynecology-obstetrics department in Dakar. Although this is a hospital prevalence, we can consider vulvovaginitis as a public health problem. Despite the higher frequency of vulvovaginitis, they are not always a reason for consultation. Indeed, in our study, they were fortuitous finding in 1/3 and as a chief complaint in 2/3 of our cases. This finding raises the problem of chronic carriage of these symptomatic infections, which may be partly responsible for the spread of STIs and HIV. In addition, the risk of gynecological and obstetric complications is higher women with vulvovaginitis. In our patients these complications were reported in the antecedents in one-fifth of the married women.

There is a variety of risk factor for infectious vulvovaginitis. Some of them tied to the host and his underlying disease, but also depend to extrinsic factors [17,18]. All these factors act by causing a disturbance of the vaginal natural defenses [6,19,20].

Intra-vaginal herbal medicine, used by a quarter of our married patients, is one of the main features of our study. While oral herbal medicine is a well-known social phenomenon in sub-Saharan Africa, intra-vaginal use is rarely reported in the literature. In our series, this practice is related to the occurrence of vaginitis du to non STI infections (p = 0.00001502) found in just over a quarter of our patients. This result is superior to that of Bohbot, et al. [21], who reported a frequency of 21.9% of bacterial vaginitis in 118 patients with infectious vaginitis. The herbs can cause an imbalance of the vaginal flora. In the literature, it is well recognized that the risk factors of bacterial vaginitis are those that disturb vaginal flora [22,23]. The other mechanism could be the introduction of an important inoculum of these germs at the same time with herbs extracts. In our series, bacterial vaginitis is also related to genito-anal sex practices (p = 0.005596). In the literature, it is shown that some vulvovaginal infections are also favored by anal penetrations preceding vaginal intercourse [24]. Similarly, in our work, bacterial vaginitis is related to lack of occupation (p = 0.01998) and pregnancy (p = 0.005216). Among the organisms that was frequently found in our study is, Group B β -hemolytic Streptococcus, which is implicated in obstetric emergencies in pregnant women.

Our work shows that the etiologies of vulvovaginitis are mixed in more than half (56.9%) of cases. However, as in the literature [5,8,15], bacterial vaginosis and candidiasis remain the most common. Among these etiologies, almost one-third of sexually active patients who had performed cervico-vaginal testing had a non-viral STI, and this same proportion of our workforce had condylomas. Regarding these non-viral STIs, trichomoniasis was the subject of several studies, but the characteristics of the studied populations varied from one study to the other, what explains the disparity of the results. In sub-Saharan Africa [13], a prevalence of trichomoniasis in women is estimated at 14%; in Côte d'Ivoire it is 8.2% [25], in Morocco among women receiving gynecological and obstetrical care, it is estimated 4.38% [26]. In Tunisia, two studies in 2004 and 2007 found a prevalence of 5% [26] and 5.37% respectively [5]. On the other hand, Diadhiou, et al. [16] found a prevalence of trichomoniasis of 3.6% in symptomatic women in gynecology-obstetrics department. These prevalences are higher than that found in a US study (2.8%) [27]. In Italy, one study found almost similar results (2%) to those found in the United States [28]. In our study, T. vaginalis was identified in 13.4% (n = 13) of cases. These results show that trichomoniasis is more common in Africa than in the Western countries. This infection is usually mild, but they increase the risk of complications in pregnant women [10].

C. trachomatis infection was found in 14.4% (n = 14) of our patients, the commonest non-viral STI in our study. This frequency is close to 10.8% of a study in Ivory coast in symptomatic women [29]. In sub-Saharan Africa [13], the prevalence of chlamydia is 7%. In obstetrics and gynecology department, in Dakar, this prevalence is estimated at 6.8% in symptomatic women [16]. In France [30], it is 3.2% in symptomatic women aged between 18 and 29 years. In the same country, Bohbot, et al. [21] reported a prevalence of 2.5% Chlamydia in 118 women with infectious vaginitis. This STI is often associated with gonorrhea, as we found in our results. This gonorrhea is found in 3% of women in sub-Saharan Africa [13]. It is 0.02 to 7.8% in pregnant women [13]. In one study in Morocco [31] in 2011, the frequency of N. gonorrheae in 797 symptomatic women was 0.5%. In the United States, in the Ginocchio, et al. [32] study, the prevalence of gonorrhea was 1.7% in 7593 symptomatic women. In France, this prevalence was 0.7% in 408 asymptomatic women [33]. In Dakar, Diadhiou, et al. [16] report a frequency of 1.6% in 192 symptomatic women. In our study, 5.6% (n = 5) of the women showed *N. gonorrheae*. The real prevalence of gonorrhea and C. trachomatis infection is difficult to determine because they are asymptomatic in 50-90% of cases [34-36]. As a result, they are often responsible for female infertility [11] and neonatal conjontivitis [14]. In fact, about 15% of salpingitis is due to gonococcus, including 5 to 8% co-infection with C. trachomatis [37,38]. Few studies show that between 10% and 40% of untreated gonococcal cervicitis develops into pelvic inflammatory disease [39]. The STIs are cofactors of HIV infection. According to some authors, the presence of an STI increases the risk of HIV transmission more than 10 times [40]. In our study, two patients diagnosed with HIV infection had an STI. One had gonorrhea and the other had condylomas. In addition, the prevalence of HIV (2%) in our STI patients is well above prevalence of 0.7% in normal papulation [41].

WHO advocates the syndromic approach for the management of vulvovaginitis, it is based on clinical findings and the appearance of vaginal discharge [42]; leucorrhoea is treated blindly without laboratory diagnosis. In our study, syndromic treatment was performed in 2/3 of the cases of vulvovaginitis. In our context, the immediate introduction of this empiric therapy with a broad-spectrum antibiotic containing antifungal and/or antibiotic [43], is justified because of delays in laboratory diagnosis, which are not compatible with a request for immediate care. In addition, the low socioeconomic level of most of our patients do not permit us to do laboratory tests for all our patients. Through this syndromic treatment (empiric therapy) we achieved a complete clinical cure of vulvovaginitis in day 28 in 68.3% (n = 69) of our patient. This rate is close to that of Zemouri, et al. [44] who found, in their systematic review, a syndromic treatment efficacy of 70%. In France, in the study by Bretelle, et al. [43], the cure rate was 82.6% in patients who received syndromic treatment and 87.6% in those who had received germ-oriented treatment, with no statistically significant difference. Although the efficacy of the syndromic treatment of vulvovaginitis is not optimal, it has a great influence on the reduction of vulvovaginal infections in Africa [45]. To improve it, it is necessary to regularly update the protocols and the drugs used in the syndromic management algorithms of vulvovaginitis in our sub-Saharan African countries.

Conclusion

Infectious vulvovaginitis is frequent. However, they are not always a reason for consultation in our working context. Although vaginal candidiasis and bacterial vaginosis are the most common cause, but the prevalence of STIs is also very high. These STIs, can cause not only gyneco-obstetric and neonatal complications but also, they are often associated with HIV. In the absence of an etiological treatment of these vulvovaginitis, empiric therapy can be improved by regular evaluations and updates.

References

- Eckert LO (2006) Clinical practice. Acute vulvovaginitis. N Engl J Med 355: 1244-1252.
- Bergogne-Bérézin E (2007) Normal vaginal flora, bacterial vaginosis, diagnosis and treatment. Antibiotiques 9: 139-144.
- 3. Rose-Marie le blanc (2009) Détecter des infections génitales basses chez la femme. Option Bio 20: 19-20.
- Judlin P (2003) Genital mycoplasmas. Gynecol Obstet Fertil 31: 954-959.
- Anane S, kaouech E, Zouari B, et al. (2010) Vulvovaginal candidiasis: Risk factors and clinical and mycological characteristics. J Mycol Med 20: 36-41.
- Benchellal M, Guelzimb K, Lemkhente Z, et al. (2011) La candidose vulvo-vaginale à l'hôpital militaire d'instruction Mohamed V (Maroc). J Mycol Med 21: 106-112.
- Sobel JD (2003) Management of patients with recurrent vulvovaginal candidiasis. Drugs 63: 1059-1066.
- 8. Krauss-Silva L, Almada-Horta A, Alves MB, et al. (2014) Basic vaginal pH, bacterial vaginosis and aerobic vaginitis: Prevalence in early pregnancy and risk of spontaneous preterm delivery, a prospective study in a low socioeconomic and multiethnic South American population. BMC Pregnancy Childbirth 14: 107.
- 9. Fleury FJ (1987) Adult vaginitis. Clin Obstet Gynecol 24: 407-438.
- 10. Alcaraz I, Duphin N, Janier M, et al. (2006) La section IST de la SFD Mycoplasmes génitaux. Ann Dermatol Venereol 133: 2S17-2S18.
- 11. Vexian RD (2009) Conduite à tenir devant une cervico-vaginite. MST 2009, 157-160.
- 12. Benchabane S, Routiot T, Muhlstein J, et al. (2011) Condylomatoses vulvovaginales et périnéales pendant la grossesse. La Revue Sage-Femme 10: 147-151.
- OMS (2007) Stratégie mondial de lutte contre les infections sexuellement transmissibles 2006-2015. Rompre la chaine de transmission.
- 14. Moore DL, MacDonald NE, Société canadienne de pédiatrie, et al. (2015) La prévention de la conjonctivite néonatale. Paediatrics & Child Health 20: 97-100.

Citation: Diop A, Joutei DA, Ndiaye MT, et al. (2020) Infectious Vulvovaginitis in Adult Women in a Hospital in Dakar. Res Rev Infect Dis 3(1):32-39

- 15. Nyirjesy P, Sobel JD (2003) Vulvovaginal candidiasis. Obstet Gynecol Clin North Am 30: 671-684.
- Diadhiou M, Ba Diallo A, Barry MS, Alavo SC, Mall I, et al. (2019) Prevalence and Risk Factors of Lower Reproductive Tract Infections in Symptomatic Women in Dakar, Senegal. Infect Dis Res Treat 12: 1-8.
- 17. Guelzim K, Lmimouni B, Kouach J, et al. (2004) Épidémiologie des candidoses vaginales à Mitrovica, Kosovo. Rev Int Serv Force Armees 77: 261-266.
- 18. Chabasse D, Guigen CI, Contet AN (1999) Mycol Med. Masson, Paris, 169.
- 19. Belec L (2002) Défenses non immunes, pré-immunes et immunes du tractus génital féminin contre les infections. J Gynecol Obstet Biol Reprod 31: 45-59.
- 20. Quan M (2000) Vaginitis: Meeting the clinical challenge. Clinical Cornerstone 3: 36-47.
- Bohbot JM, Sednaou P, Verriere F, et al. (2012) The etiologic diversity of vaginitis. Gynécologie obstétrique & Fertilité 40: 578-581.
- Wilson JD, Lee RA, Balen AH, et al. (2007) Bacterial vaginal flora in relation to changing oestrogen levels. Int J STD AIDS 18: 308-311
- 23. Parks DK, Yetman RJ, Moyer V, et al. (2000) Early-onset neonatal group B streptococcal infection: Implications for practice. J Pediatr Heal Care 14: 264-269.
- 24. Rylander E, Berglund Al, Krassny C, et al. (2004) Vulvovaginal candida in a young sexually active population: Prevalence and association with orogenital sex and frequent pain at intercourse. Sex Transmit Infect 80: 54-57.
- 25. Faye-Kette YH, Kouassi AA, Sylla-Koko DF, et al. (1993) Prevalence of 4 agents of sexually transmitted diseases in leukorrhea in Abidjan (Ivory Coast). Bull Soc Pathol Exot 86: 245-247.
- 26. Zribi M, Ben MK, Abid F, et al. (2008) Syndromic approach to sexually transmitted infections in Tunisian women: Bacteriological validation. Int J STD AIDS 19: 112-114.
- 27. Miller WC, Swygard H, Hobbs MM, et al. (2005) The prevalence of Trichomoniasis in young adults in the United States. Sex Transm Dis 32: 593-598.
- 28. Spinillo A, Bernuzzi AM, Cevini C, et al. (1997) The relationship of bacterial vaginosis, Candida and Trichomonasinfection to symptomatic vaginitis in postmenopausal women attending avaginitis clinic. Maturitas 27: 253-260.
- 29. Bankolé H, Faye-Ketté H, Laruche G, et al. (2001) Etude par culture cellulaire de l'infection active due à chlamydiae trachomatis dans une population de femmes symptomatiques à Abidjan. Bull Soc Pathol Exot 94: 235-238.
- 30. Bébéar C, Cazanave C (2009) Mycoplasmes urogénitaux. MST 2009, 57-61.
- 31. (2011) Direction de l'Épidémiologie et de Lutte contre les Maladies (ministère de santé Maroc) Programme National de Lutte contre les IST/SIDA Rabat: DELM.

- 32. Ginocchio CC, Chapin K, Smith JS, et al. (2012) Prevalence of Trichomonas vaginalis and coinfection with Chlamydia trachomatis and Neisseria gonorrhoeae in the United States as determined by the Aptima Trichomonas vaginalis nucleic acid amplification assay. J Clin Microbiol 50: 2601-2608.
- 33. Toyer AL, Trignol VN, Mereghetti L, et al. (2012) Interest of simultaneous Chlamydia trachomatis and Neisseria gonorrhoeae screening at the time of preabortion consultation. Contraception 86: 572-576.
- 34. Cedef (2012) Item 95-Maladies sexuellement transmissibles : infections urogénitales à gonocoque et Chlamydia trachomatis (en dehors de la maladie de Nicolas-Favre). Ann Dermatol Venereol 139: A57-A61.
- 35. Duplin N, Janier M, Bouscarat F, et al. (2006) Chlamydia trachomatis infection. Ann Dermatol Venereol 133: 2513-2514.
- Geniaux M, Vaillant L, Morel P, et al. (2002) Sexually transmitted diseases. Gonococcus and Chlamydia trachomatis urogenital infections (except Nicolas Favre's disease). Ann Dermatol Venereol 129: S65-S68.
- 37. Chen JZ, Gratrix J, Smyczek P, et al. (2018) Gonococcal and chlamydial cases of pelvic inflammatory disease at 2 canadian sexually transmitted infection clinics, 2004 to 2014: A retrospective cross-sectional review. Sex Transm Dis 45: 280-282.
- 38. Ness RB, Soper DE, Holley RL, et al. (2002) Effectiveness of in patient and out patient treatment strategies for women with pelvic inflammatory disease: Results from the pelvic inflammatory disease evaluation and clinical health (PEACH) randomized trial. Am J Obstet Gynecol 186: 929-937.
- 39. Tarr ME, Gilliam ML (2008) Sexually transmitted infections in adolescent women. Clin Obstet Gynecol 51: 306-318.
- 40. Ramjee G, Moonsamy S, Samantha AN, et al. (2016) Individual and population level impact of key HIV risk factors on HIV incidence rates in Durban, South Africa. PLoS One 11: e0153969.
- 41. ONUSIDA République du Sénégal. Rapport de situation sur la riposte nationale à l'épidémie de VIH/SIDA Sénégal: 2010-2011-2012.
- 42. Strobel M (2004) Infections sexuellement transmissibles.
- 43. Bretelle F, Chiarelli P, Palmer I, et al. (2015) Prise en charge des infections vaginales après échec d'un traitement probabiliste: Le prélèvement vaginal est-il réellement utile? Gynécol Obstet & Fertilité 43: 139-143.
- 44. Zemouri C, Wi TE, Kiarie J, et al. (2016) The performance of the vaginal discharge syndromic management in treating vaginal and cervical infection: A systematic review and meta-analysis. PLoS One 11: e0163365.
- 45. Johnson LF, Dorrington RE, Bradshaw D, et al. (2011) The effect of syndromic management interventions on the prevalence of sexually transmitted infections in South Africa. Sex Reprod Healthc 2: 13-20.

DOI: 10.36959/719/560

Copyright: © 2020 Diop A, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

